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		"2,3-f"))		

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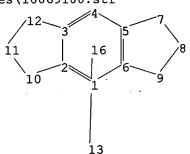
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chain nodes:

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ring nodes :

1 2 3 4 5 6 7 8 9 10 11 12

ring bonds :

1-2 1-6 2-3 2-10 3-4 3-12 4-5 5-6 5-7 6-9 7-8 8-9 10-11 11-12

exact/norm bonds :

2-10 3-12 5-7 6-9 10-11 11-12

exact bonds :

7-8 8-9

normalized bonds :

1-2 1-6 2-3 3-4 4-5 5-6

Match level:

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9.6% PROCESSED 1000 ITERATIONS INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED) SEARCH TIME: 00.00.01

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16 TO

1 SEA SSS SAM L1 L2

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1 ANSWERS REGISTRY COPYRIGHT 2003 ACS on STN L2

2H-Furo[2,3-f]indole, 3,5,6,7-tetrahydro-2,2,4,8-tetramethyl-3-(4-IN methylphenyl) - (9CI)

C21 H25 N O MF

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

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ALL ANSWERS HAVE BEEN SCANNED

=> s l1 full FULL SEARCH INITIATED 11:06:29 FILE 'REGISTRY' FULL SCREEN SEARCH COMPLETED - 206535 TO ITERATE

100.0% PROCESSED 206535 ITERATIONS

32 ANSWERS

SEARCH TIME: 00.00.03

32 SEA SSS FUL L1 . L3

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FULL ESTIMATED COST

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7 L3 L4

=> s wo2001014384?/pn

1 WO2001014384?/PN L5

(WO2001014384/PN)

=> s 14 not 15

6 L4 NOT L5

=> d 1-6 cbib pi abs hitstr

ANSWER 1 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN Document No. 137:262696 The silver nitrate oxidation of 2,2,4,6,7-pentamethylcoumaran-5-ol. Schadel, Uta; Gruner, Margit; Habicher, Wolf D. (Institute of Organic Chemistry, Dresden University of Technology, Dresden, 01069, Germany). Tetrahedron, 58(25), 5081-5086 (English) 2002. CODEN: TETRAB. ISSN: 0040-4020. Publisher: Elsevier

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The silver nitrate oxidation of 2,2,4,6,7-pentamethylcoumaran-5-ol was investigated. The complex mixture of products formed is in partial disagreement with the mechanisms supposed so far and suggests a less strong Mills-Nixon effect than assumed until now. Consecutive reactions of 2,2,4,7-tetramethylcoumaran-5,6-dione, which was formed as main product, were examined

IT 462092-02-0P

RL: SPN (Synthetic preparation); PREP (Preparation) (mechanism of silver nitrate oxidation of 2,2,4,6,7-pentamethylcoumaran-5-ol and further reactions of 2,2,4,7-tetramethylcoumaran-5,6-dione as its main oxidation product)

RN 462092-02-0 CAPLUS

CN Furo[2,3-b]phenazine, 2,3-dihydro-2,2,4,11-tetramethyl- (9CI) (CA INDEX NAME)

L6 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN
1994:298644 Document No. 120:298644 Preparation of furo- or pyranoquinoline
derivatives or their salts as cardiotonics, antiarrhythmics, and
vasodilators. Kyotani, Yoshinori; Taima, Tsutomu; Kurihara, Juji;
Kitamura, Takahiro; Kamya, Kazuhiro; Yamaguchi, Takashi; Onoki, Kazuhiro;
Sato, Seiichi; Oota, Tomio; Uchida, Yasuyoshi (Kowa Co, Japan). Jpn.
Kokai Tokkyo Koho JP 05339271 A2 19931221 Heisei, 14 pp. (Japanese).
CODEN: JKXXAF. APPLICATION: JP 1992-145545 19920605.

PATENT NO. KIND DATE APPLICATION NO. DATE

----PI JP 05339271 A2 19931221 JP 1992-145545 19920605
JP 3153335 B2 20010409

GI

AB The title derivs. I [R1-2 = H, lower alkyl; R3 = (un)substituted lower alkyl, lower alkanoyloxy, OH, lower alkylsulfonyloxy, azido, amino; A = O, direct bond; when A = O then B = direct bond or CH:CH; when A = direct

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bond then B = O] or their salts are prepared as cardiotonics, antiarrhythmics, and vasodilators (no data). A solution of 7-acetoxy-1,2-dihydro-6-(2,3-epoxypropyl)-8-methylquinolin-8-one (preparation from 3-amino-o-cresol in 6 steps) in DMF was treated with aqueous NaOH at 50° for 30 min to give 61.8% 2-hydroxymethyl-9-methyl-2,3,7,8-tetrahydrofuro[3,2-g]quinolin-7-one.

IT 154521-24-1P 154521-25-2P 154521-26-3P

154521-27-4P 154521-28-5P

RL: SPN (Synthetic preparation); PREP (Preparation) (preparation of, as cardiotonic and antiarrhythmic and vasodilator)

RN 154521-24-1 CAPLUS

CN Furo[2,3-g]quinolin-6(5H)-one, 2,3-dihydro-2-(hydroxymethyl)-4,9-dimethyl-(9CI) (CA INDEX NAME)

O
$$\stackrel{\text{H}}{\underset{\text{N}}{\bigvee}}$$
 $\stackrel{\text{Me}}{\underset{\text{CH}_2-\text{OH}}{\bigvee}}$ $\stackrel{\text{CH}_2-\text{OH}}{\underset{\text{Me}}{\bigvee}}$

RN 154521-25-2 CAPLUS

CN Furo[2,3-g]quinolin-6(5H)-one, 2,3-dihydro-4,9-dimethyl-2-[[(methylsulfonyl)oxy]methyl]- (9CI) (CA INDEX NAME)

$$\begin{array}{c|c} O & Me & O & O \\ H & Me & CH_2-O-S-Me \\ \hline Me & O & O \\ \end{array}$$

RN 154521-26-3 CAPLUS

CN Furo[2,3-g]quinolin-6(5H)-one, 2-(azidomethyl)-2,3-dihydro-4,9-dimethyl-(9CI) (CA INDEX NAME)

$$\begin{array}{c|c}
Me \\
N \\
N \\
Me
\end{array}$$

$$\begin{array}{c}
CH_2 - N_3 \\
Me
\end{array}$$

RN 154521-27-4 CAPLUS

CN Furo[2,3-g]quinolin-6(5H)-one, 2-(aminomethyl)-2,3-dihydro-4,9-dimethyl-(9CI) (CA INDEX NAME)

O
$$H$$
 CH_2-NH_2 O Me Me

RN 154521-28-5 CAPLUS

CN Furo[2,3-g]quinolin-6(5H)-one, 2-(aminomethyl)-2,3-dihydro-4,9-dimethyl-, monohydrochloride (9CI) (CA INDEX NAME)

O
$$\frac{H}{N}$$
 CH_2-NH_2 Me

HCl

L6 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN

1993:230163 Document No. 118:230163 Studies on the constituents of Aristolochia liukiuensis. II. Kazuhito, Ogihara; Zhao, Jiaping; Higa, Matsutake; Yogi, Seiichi (Coll. Sci., Univ. Ryukyus, Nishihara, 903-01, Japan). Bulletin of the College of Science, University of the Ryukyus, 54, 17-28 (Japanese) 1992. CODEN: BCSRDZ. ISSN: 0286-9640.

AB The root of Aristolochia liukiuensis contained aristolactone, mansonone G, dehydrooxoperezinone, aristololactam DII, 3,4-methylenedioxy-8-methoxyphenanthrene-1-carboxylic acid, aristolochic acid II, and aristolochic acid IV Me ester.

IT 18142-22-8P

RL: SPN (Synthetic preparation); PREP (Preparation)
 (preparation of)

RN 18142-22-8 CAPLUS

CN 4H-Isobenzofuro[7,1-ab]phenazine, 3-methoxy-1,4,4,6-tetramethyl- (8CI, 9CI) (CA INDEX NAME)

GI

L6 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN

1987:18852 Document No. 106:18852 Oxygenation of cadalene and eudalene in polar aprotic solvents. Takekuma, Shinichi; Matsubara, Yoshiharu; Yamamoto, Hiroshi; Nozoe, Tetsuo (Fac. Sci. Technol., Kinki Univ., Higashi-Osaka, Japan). Yukagaku, 34(12), 1026-8 (English) 1985. CODEN: YKGKAM. ISSN: 0513-398X. OTHER SOURCES: CASREACT 106:18852.

AB Oxygenation of cadalene (I, R=R1=Me) in DMSO or DMF at 120° for 30 h yielded I (R = CH2OH, CHO, R1 = Me; R = Me, R1 = CHO), quinone II, and naphthofuran III. Similar oxidation of eudalene (IV, R2 = Me, R3 = CHMe2) gave IV (R2 = Me, CH2OH, CHO, R3 = CHMe2; R2 = Me, R3 = Ac).

RN 10124-06-8 CAPLUS

CN 4H-Isobenzofuro[7,1-ab]phenazine, 1,4,4,6-tetramethyl- (7CI, 8CI, 9CI) (CA INDEX NAME)

Page 8

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN

1983:125923 Document No. 98:125923 Versatile syntheses of quinolines by annulation of pyridines. Synthesis of furo[2,3-g]- and -[3,2-g]quinolines. Ghera, E.; Ben-David, Y.; Rapoport, H. (Dep. Org. Chem., Weizmann Inst. Sci., Rehovot, Israel). Journal of Organic Chemistry, 48(6), 774-9 (English) 1983. CODEN: JOCEAH. ISSN: 0022-3263. OTHER SOURCES: CASREACT 98:125923.

GI

An ew, versatile annulation route for the synthesis of substituted quinolines has been developed by using regioisomeric bifunctional pyridine derivs. with vicinal bromomethyl and (phenylsulfonyl)methyl groups. The sequence consists of (a) alkylation of substituted di-Et malonates with these bromomethylpyridines and (b) intramol. acylation with concomitant decarboxylation and leads to quinoline derivs. variously substituted in the carbocycle. A simultaneous desulfurization-aromatization of the carbocycle has been developed for these cyclized sulfones.

5-(Phenylsulfonyl)-7-allyl-6-quinolinol, obtained via this cyclization and dehydrogenation, was then used for the preparation of furo[2,3-g]quinoline derivs. The novel parent systems furo[2,3-g]- and -[3,2-g]quinoline (I and II) were obtained in good yield in a one-operation acid-induced cyclization-elimination sequence from the bicyclic annulation products III and IV, resp.

IT 84583-45-9P

RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)

(preparation and dehydration of)

RN 84583-45-9 CAPLUS

CN Furo[2,3-g]quinolin-2-ol, 2,3-dihydro-9-(phenylsulfonyl)- (9CI) (CA INDEX NAME)

IT 84583-48-2P

RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)

(preparation and dehydroiodination of)

RN 84583-48-2 CAPLUS

CN Furo[2,3-g]quinoline, 2,3-dihydro-2-(iodomethyl)-9-(phenylsulfonyl)- (9CI) (CA INDEX NAME)

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN

1968:114314 Document No. 68:114314 Contribution to the chemistry of perezone. Joseph-Nathan, Pedro; Reyes, J.; Gonzalez, Maria P. (Inst. Politec. Nac. Mexico City, Mexico City, Mex.). Tetrahedron, 24(10), 4007-13 (English) 1968. CODEN: TETRAB. ISSN: 0040-4020.

GI For diagram(s), see printed CA Issue.

AB The sequence of reactions described confirms that perezinone (I) is a quinone methide related to the 2H-naphtho[1,8-bc]furan system. The structures of two oxidation derivs. isolated earlier are established.

IT 18142-22-8P

RL: SPN (Synthetic preparation); PREP (Preparation)
 (preparation of)

RN 18142-22-8 CAPLUS

CN 4H-Isobenzofuro[7,1-ab]phenazine, 3-methoxy-1,4,4,6-tetramethyl- (8CI, 9CI) (CA INDEX NAME)

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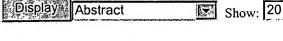
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Lipid peroxidation in human proteinuric disease.

Solin ML, Ahola H, Haltia A, Ursini F, Montine T, Roveri A, Kerjaschki Holthofer H.

The Haartman Institute, Division of Bacteriology and Immunology, Universi Helsinki, Helsinki, Finland.

BACKGROUND: While metabolically generated oxidants are produced loca experimental glomerular diseases, little is still known of their significance an respective scavenger systems in human glomerular diseases. METHODS: He studied kidneys from patients with congenital nephrotic syndrome of the Fini type (CNF), a human model disease of isolated proteinuria. Expression of spe mRNAs for a major antioxidant system against lipoperoxidation [phospholip hydroperoxide glutathione peroxidase (PHGPx)] and for mitochondrial prote were studied in Northern blotting together with analysis of PHGPx in semiquantitative reverse transcription-polymerase chain reaction (RT-PCR). respective proteins and lipoperoxide (LPO) adducts malonyldialdehyde (MD and 4-hydroxynonenal (4-HNE) were analyzed in immunohistochemistry. RESULTS: PHGPx and the mitochondrially encoded subunits of cytochrome oxidase were distinctly down-regulated within the glomeruli of CNF kidneys These changes were confirmed in semiquantitative RT-PCR. Increases of lipoperoxidation products MDA and 4-HNE were constantly found in the glomeruli of CNF. In agreement with findings in CNF, similar results were obtained in biopsies from other human glomerular diseases. CONCLUSION! These findings suggest that local mitochondrial damage initiates LPO, which causes deposition of the cytotoxic LPO products in glomeruli, as seen especi-CNF kidneys. Together with down-regulation of the local antioxidant protect these may be important pathophysiologic mechanisms in human glomerular disease.

PMID: 11168930 [PubMed - indexed for MEDLINE]



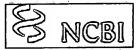




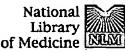












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Combination of a calcium antagonist, a lipid-peroxidation inhib and an angiotensin AT1-receptor antagonist provides additive myocardial infarct size-limiting effect in pigs.

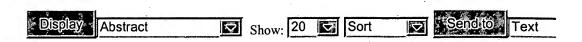
Shimizu M, Wang QD, Sjoquist PO, Ryden L.

Department of Cardiology, Karolinska Hospital, Stockholm, Sweden.

The calcium antagonist felodipine, the lipid-peroxidation inhibitor H290/51, the angiotensin II type 1 (AT1)-receptor antagonist candesartan all exert bene effects on myocardial ischemia/reperfusion injury. This study was undertaken test the hypothesis that a combination of these drugs with different pharmaco properties could exert additive cardioprotective effects. Anesthetized pigs we subjected to 45 min of left anterior descending coronary artery occlusion foll by 240 min of reperfusion. Five groups of pigs were randomly given either 0 microM (7 nmol/kg) felodipine, 1.0 microM (3.1 microg/kg) H 290/51, 4.2 microM (20 microg/kg) candesartan, a cocktail of these three drugs, or vehic = 6 for each) for 30 min starting at 5 min before reperfusion by coronary ven retroinfusion, which delivers drugs specifically to the ischemic regions. Syste segment shortening (%SS) was measured by sonomicrometer. The myocardia at risk and the final infarct size were determined by Evans blue and 2,3,5-trip tetrazolium chloride staining. The hemodynamics did not change significantl during the study. In the vehicle group, the recovery of coronary flow was not maintained during reperfusion, and it was significantly lower after 240 min o reperfusion than during the preischemic period (p < 0.05). The coronary flow the drug-treated groups was approximately the same by the end of the reperfi period as that before the induction of ischemia. In the ischemic myocardium, slightly recovered during reperfusion in the four drug-treated groups, but not vehicle group. The infarct size, expressed as a percentage of the myocardial a risk, was smaller in all four drug-treated groups compared with the vehicle gr The infarct size in the cocktail group was significantly smaller than that in th groups given felodipine, H290/51, or candesartan alone. These results demor that a combination of a calcium antagonist, a lipid-peroxidation inhibitor, and angiotensin AT1-receptor antagonist has an additive effect on infarct limitative indicating that combined therapy with agents having different pharmacologic modes of action may provide better cardioprotection than any of the drugs al-The findings also support the view that reperfusion injury is possibly mediate

a combination of mechanisms.

PMID: 10511125 [PubMed - indexed for MEDLINE]



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☐ 1: Eur J Anaesthesiol. 1996 May; 13(3):279-89.

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Efficacy and mechanisms of action of the cytoprotective lipid peroxidation inhibitor tirilazad mesylate in subarachnoid haemorrhage.

Hall ED.

CNS Diseases Research, Pharmacia & Upjohn, Inc., Kalamazoo, Michigan 4 USA.

Subarachnoid haemorrhage (SAH) following cerebral aneurysm rupture or tr can result in the induction of secondary ischaemic brain damage via a decrea microvascular perfusion, a disruption of the blood-brain barrier and conseque vasogenic oedema, and the delayed spasm of the major cerebral arteries (i.e. vasospasm). It is increasingly apparent that oxygen radical-induced, iron-cata lipid peroxidation (LP) within the subarachnoid blood and vascular wall play key role in the occurrence of these secondary events. Tirilazad mesylate is a properties of LP that works by a combination of radical scavent and membrane stabilizing properties. It has been demonstrated to attenuate the acute and delayed vascular consequences of SAH and to protect the brain againschaemic insults. Much of its action is mediated by an effect on the vascular endothelium, although it also appears to exert some direct neuroprotection ar inhibit LP in the subarachnoid blood. These actions of tirilazad in experimen SAH are reviewed.

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PMID: 8737119 [PubMed - indexed for MEDLINE]

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Aminosteroids for acute traumatic brain injury.

Roberts I.

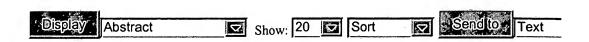
Department of Epidemiology, Institute of Child Health, 30 Guilford Street, London, UK, WC1N 1EH. Ian.roberts@ich.ucl.ac.uk

BACKGROUND: Traumatic brain injury is a leading cause of premature dea and disability. Post-traumatic membrane lipid peroxidation has been propose one mechanism leading to secondary brain damage following head injury. Aminosteroids have been shown to inhibit lipid peroxidation in laboratory an and have the potential to improve outcome following head injury. OBJECTI' To quantify the effectiveness and safety of aminosteroids in the treatment of traumatic brain injury. SEARCH STRATEGY: We searched the Cochrane Ir Group trials register, The Cochrane Controlled Trials Register, MEDLINE at EMBASE. We contacted experts in the field and the company that manufacti tirilazad. SELECTION CRITERIA: We sought to identify all randomised controlled trials of aminosteroids versus placebo in the treatment of acute traumatic brain injury. Studies using a quasi random form of allocation, such alternation, were excluded from the review. DATA COLLECTION AND ANALYSIS: One reviewer examined the electronic search results for reports possibly relevant trials for retrieval in full. Two reviewers (IR and PA) applic selection criteria independently to the trial report, with no disagreement. MA RESULTS: Two randomised controlled trials have examined the effect of the aminosteroid tirilazad mesylate on death and disability following head injury date, only the results of one of these trials are available for analysis. The risk death in patients treated with tirilazad was almost identical to those given pla RR=1.05 (95% confidence interval 0.86 to 1.29). The risk of death and sever disability in patients treated with tirilazad was again almost identical to those given placebo RR=1.07 (95% confidence interval 0.93 to 1.23). REVIEWER CONCLUSIONS: There is no evidence to support the routine use of aminost in the management of traumatic head injury. On the basis of the existing evid from randomised trials of aminosteroids in head injury it is not possible to rethe possibility of moderate but potentially clinically important benefits or har further randomised controlled trial of tirilazad mesylate with 1156 participan been completed, the results of which should become available in the near futi

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